NEGATIVE PRESSURE DRESSINGS FOR OPEN FRACTURE WOUNDS

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TRADITIONAL MANAGEMENT OF THE OPEN FRACTURE WOUND

The wound associated with an open fracture is commonly underestimated in its representation of the underlying soft tissue injury. The traumatic injury to the periosteum, muscle, fascia, and subcutaneous tissue is often greater than expected, especially with a crushing mechanism of injury. The open fracture wound evolves over time, and necrotic tissue becomes more evident. The degree of exposure of the underlying structures (bone, tendon, etc.) may change as the wound evolves. Often a wound seen at the time of injury is thought to be amenable to delayed primary closure. Yet after several debridements and internal fixation of the fracture, the effects of altered limb shape and soft tissue swelling change the wound, making more advanced techniques in soft tissue coverage necessary.

The management of an open fracture is often difficult. The mainstay of treatment involves timely initial debridement and irrigation of the wound. This is repeated every 48 to 72 hours until no further contamination or necrotic tissue remains in the wound. Traditional instruction has been to leave at least the traumatic portion of the wound open, to allow for fluid and bacterial egress from the wound bed. When the wound shows no further signs of tissue necrosis, definitive soft tissue coverage can be performed. This can be achieved with delayed primary closure of the wound, split-thickness skin graft over a vascularized bed, and for the more severe injuries, rotational or microvascular tissue transfers.[4]

This poses the question of how to best manage the open fracture wound between debridements, and prior to definitive soft tissue coverage. The ideal open fracture wound management system would prevent desiccation of exposed vital structures, promote vascular ingrowth, remove edema from the wound bed, limit bacterial proliferation, minimize pain, prevent repetitive trauma to the wound bed, and prevent secondary bacterial contamination from the environment. Traditional methods of managing the open fracture wound consist of simple dressing changes, skin substitutes, and antibiotic-impregnated bead pouches.

Traditional wet-to-dry dressing changes have been employed using a variety of solutions, including dilute povidone-iodine, dilute bleach, antibiotics, and normal saline. A moistened dressing is applied to the wound, and allowed to dry. With removal of the dry dressing, necrotic tissue is removed with the dressing. Dry dressings can be applied to wounds with more exudate. Dressing changes have the advantage of universal availability of supplies. The disadvantages include desiccation of exposed structures, maceration of skin in wounds with significant effluent, repeated wound trauma, and discomfort with the procedure. The greatest disadvantage is the vast opportunity for secondary contamination of the wound with repeated exposure to the environment. This is likely a significant factor in post-traumatic infections.

Little information is available on the use of skin substitutes for temporary management of open fracture wounds. Skin substitutes include xenografts, human allografts, and a variety of synthetic membranes. Xenograft and allograft can be placed over a clean wound bed, and will adhere to viable tissue. Both prevent desiccation of the wound, limit bacterial proliferation, decrease wound pain, and protect underlying structures. Synthetic adherent dressings are available containing an inner layer of collagen, which can bind to a clean wound bed, and an outer synthetic layer which is impermeable to bacteria. The tight adherence decreases exudate, and protects underlying structures from desiccation. Unlike skin, synthetic membranes can be stored at room temperature and have a longer shelf-life. Unfortunately, these substances can be expensive, and are not universally available. In addition, with removal of these substances, bits of collagen may remain in the wound bed and become a source of chronic inflammation.

An antibiotic-impregnated bead pouch is a reliable method for managing an open fracture wound. Certain antibiotics, particularly tobramycin, can be mixed with

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polymethylmethacrylate during its polymerization, and formed into small spheres. The antibiotic will leach out of the beads, resulting in bactericidal levels in the surrounding tissue.[3] Henry et al. described the technique of a bead pouch, where antibiotic bead chains were placed in an open fracture wound, and covered with an occlusive dressing.[9] The advantage of this technique is reduced bacterial contamination in the wound. Not only is the wound bathed in high concentrations of antibiotic, but the fracture site is sealed from the environment, reducing outside sources of contamination. When used as an adjuvant to debridement and parenteral antibiotics, use of the bead pouch has been shown to decrease the infection rate in open fractures.[11,15] The main disadvantage of this technique is the time-consuming nature of antibiotic bead production in the operating room. Some have advocated the use of premade chains of antibiotic beads to hasten the procedure.[6] It is also difficult to obtain a good seal with the occlusive dressing, particularly with large wounds or wounds around external fixator pins. An incomplete occlusive dressing allows leakage of the antibiotic-rich fluid, skin maceration, and potential exposure of the wound to secondary bacterial contamination.

NEGATIVE PRESSURE DRESSINGS

Negative pressure dressings are an attractive option for the interim management of open fracture wounds. The negative pressure dressing consists of a polyurethane ether foam sponge that is cut to fit the contours of the wound. Into the sponge is placed a noncollapsable evacuation tube. The evacuation tube is connected to a canister that collects the effluent. The canister is coupled to the control box, which regulates the force applied through the dressing. The dressing is sealed with an occlusive drape (Figure 3). The typical setting is 125 mm subatmospheric pressure for the first 48 hours, then intermittent mode (5 minutes on, 2 minutes off) for the remaining duration of therapy. Clinical studies demonstrated that wound healing is accelerated using this cyclic mode (1). The dressing is changed every 48 to 72 hours, to prevent aggressive ingrowth of granulation tissue into the sponge. This correlates well with the timing of debridements.

Prior experience with negative pressure dressings is encouraging. Chronic wounds caused by pressure, venous stasis, radiation, diabetes, and vasculitis have been successfully treated with negative pressure dressings. Therapy continues until the wound heals or the granulating bed can be treated with a lesser procedure, such as split-thickness skin grafting.[1] Dehisced and infected surgical wounds in the chest and abdomen have been treated with negative pressure dressings. Again, therapy is continued until a split-thickness skin graft can be placed over the bed.[1,13] This is a great advantage for patients who are often poor candidates for traditional wound management strategies such as free microvascular tissue transfers. Acute wounds such as large soft tissue avulsions and gunshot wounds have been managed successfully with negative pressure dressings.[1]

Negative pressure dressings have also been shown to be a useful adjuvant in the management of wounds associated with limb threatening injuries. A group of patients with either failed free flap coverage, wounds too large to adequately cover with free flaps, or infected wounds waiting for free flap coverage were treated with negative pressure dressings. Although some needed additional surgical procedures, no amputations occurred and adequate soft tissue coverage was obtained in all patients.[10]

There are several advantages negative pressure dressings have for acute open fracture wounds. First, the occlusive dressing completely isolates the wound, decreasing the risk of secondary contamination from the environment. Second, the negative pressure dressing removes edema from the limb. Edema present in the wound bed increases the pressure in the tissue, slowing capillary inflow, and impeding venous and lymphatic drainage. Removal of edema by the negative pressure dressing improves capillary blood flow, which increases the delivery of oxygen and nutrients to the wound.[1,16,18] Removal of edema fluid also removes compounds that are detrimental to wound healing. Factors removed from chronic wound fluid have been found to suppress the proliferation of keratinocytes, fibroblasts, and vascular endothelial cells.[1,2,7] Elevated levels of proteases (e.g. collagenase, elastase) and their degradation products are also found in this fluid.[1,8,19,20] Third, bacterial proliferation is limited. The dressing results in capillary ingrowth into the wound. This increase in vascularity provides the oxygen and immune cells that antagonize bacterial growth. Although the wound remains colonized, wounds treated with negative pressure dressings show a decrease in the bacterial load after 3-4 days. Quantitative cultures have demonstrated bacterial colonization in the range of 10^2-10^3 organisms per gram of tissue. Successful wound healing correlates with bacterial counts less than 10^5 organisms per gram of tissue.[1,12] Finally, the application of mechanical force to a wound produces a favorable milieu. Negative pressure applied to a wound results in an increased rate of cell mitosis, new blood vessel formation, and recruitment of adjacent cells.[1,5,14,17] In this favorable environment, is not uncommon for the growth of granulation tissue to be so aggressive that a lesser surgical procedure, such as a split-thickness skin graft, can be utilized instead of a more taxing procedure, such as a microvascular tissue transfer, to achieve soft tissue cov-

erage. The main disadvantage of this technique is the requirement for special equipment. There is also a significant learning curve in applying the dressing, especially with large wounds, multiple wounds, and wounds around external fixator pins.

Complications of negative pressure dressing treatment can occur. Some patients complain about pain with initiation of negative pressure to the wound. This usually abates over time. Most other problems are related to improper dressing application. Erythema around wound edges can be seen with a dressing that extends to the intact skin. This erythema can be mistaken for cellulitis, but really represents hyperemia, and will abate quickly with discontinuation of therapy. Necrosis of the skin around the evacuation tube has been observed if the occlusive drape is applied too tightly in this region. Ingrowth of granulation tissue into the sponge has been observed, most commonly if the dressing has been in place over 72 hours.[1]

CASE EXAMPLE

A 42 year-old man was a pedestrian struck by a car. His injuries included an open proximal humerus fracture and an open proximal tibial fracture. The proximal tibial fracture was typical for a bumper strike injury—a comminuted proximal metaphyseal injury with articular extension, and a significant soft tissue injury. The proximal tibial fracture was associated with a 1 cm wound on the medial proximal tibia. On the night of injury, the open fracture wound was extended, and the wound and the fracture debrided (Figure 1). A few fragments of devitalized bone were removed. No gross contamination was found in the wound. The wound extensions were closed easily, leaving the small traumatic wound open. The leg was stabilized by placing a bridging external fixator across the knee. Forty-eight hours later, the patient returned to the operating room for a repeat debridement of the open fracture. Epidermolysis of the wound edges was observed (Figure 2). After debridement, the wound edges could not be approximated, so a Vacuum Assisted Closure (VAC) dressing (Kinetic Concepts, Inc., San Antonio, TX) was applied to the open wound, and set at 125 mm continuous subatmospheric pressure (Figure 3). Consultation with the plastic surgery service was made for timing of definitive soft tissue coverage. Forty-eight hours later, the patient returned to the operating room. The open fracture was debrided, and no further necrotic tissue was present. Definitive internal fixation of the fracture was performed (Figures 4A and 4B). A VAC was applied to the medial wound, and set at 125 mm subatmospheric pressure in the intermittent mode. Forty-eight hours later, the patient returned to the operating room where

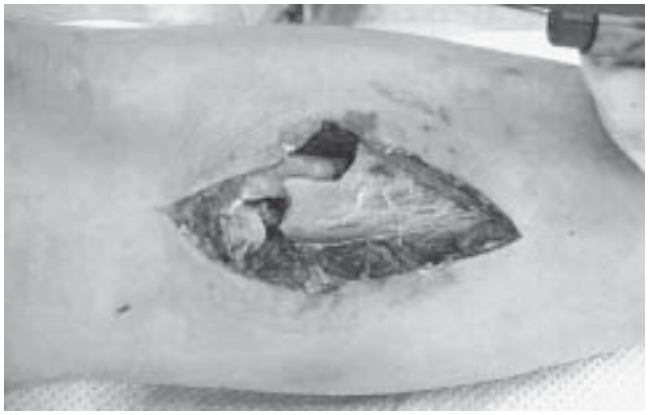


Figure 1. The open fracture wound. Significant skin contusion and extensive periosteal stripping are demonstrated.

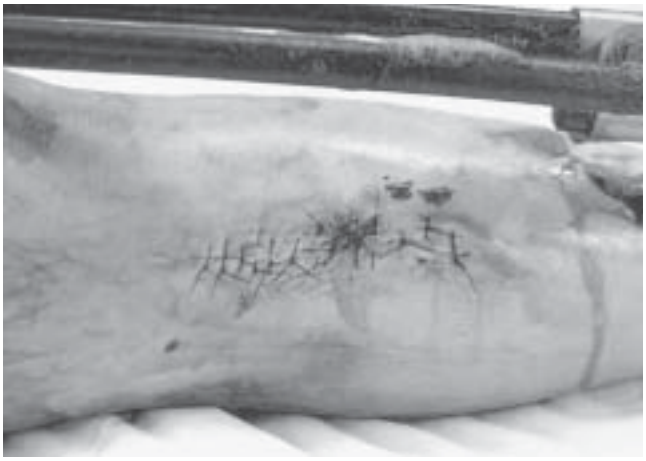


Figure 2. The wound before the second debridement. The wound extensions have been closed. The limb is edematous. The skin in the center of the wound has questionable viability.

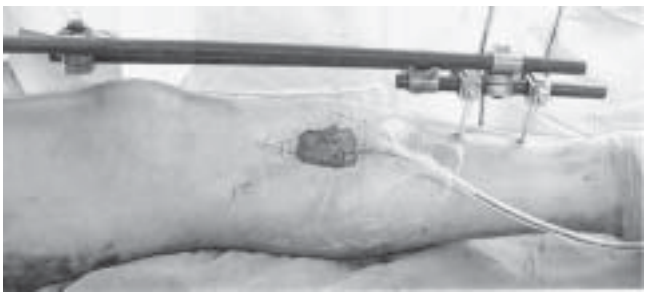
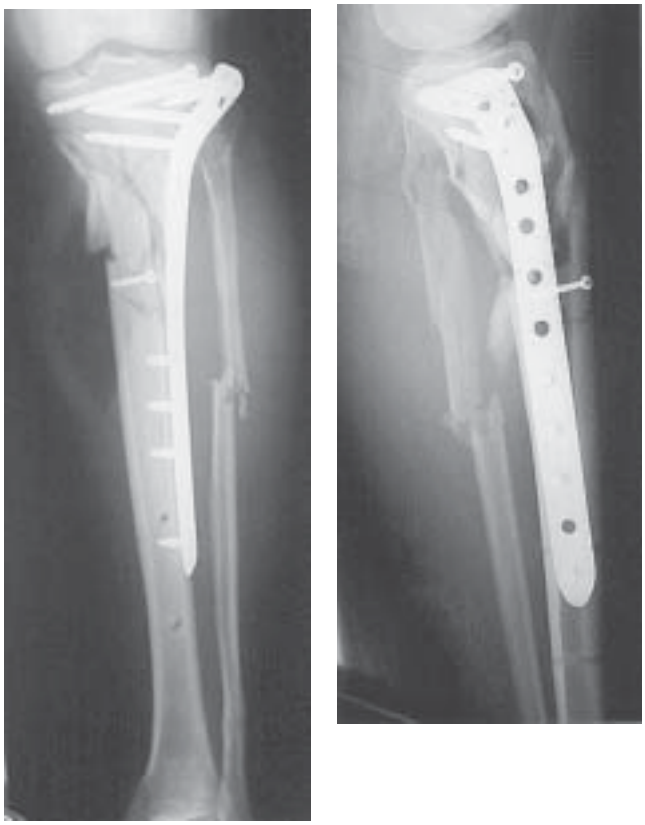


Figure 3. The negative pressure dressing has been applied to the open fracture wound. The sponge has been cut to fit the contours of the wound. An evacuation tube connects the sponge to a source of constant subatmospheric pressure. An occlusive drape seals the dressing.

a medial gastrocnemius rotational flap and split-thickness skin graft was performed to close the soft tissue envelope. One month later, all wounds were completely healed and edema was minimal (Figure 5). Twelve weeks after injury, the articular fracture lines had healed, and enough callus was present in the metaphyseal region to begin weightbearing. No evidence of infection was present.



Figures 4A and 4B. AP and lateral radiographs of the tibia demonstrate the skeletal injury. The articular fracture is reduced and held with a lag screw. The comminuted metaphyseal fracture is stabilized by a less invasive stabilization system plate (Synthes, Paoli, PA).

Figure 5. Clinical appearance of the leg one month after medial gastrocnemius rotational flap and split-thickness skin graft. The knee is to the left and the ankle is to the right. The soft tissue envelope is completely healed.

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